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TRAUMATIC AFFECTIONS OF THE CERVICAL REGION OF THE SPINAL CORD, SIMULATING SYRINGOMYELIA.[1]

By JAMES HENDRIE LLOYD, A.M., M.D.,
Physician to the Philadelphia Hospital.

THE earlier writers, of what may be called the present epoch of clinical neurology, recognized and described a particular type of lesion of the spinal cord which was localized in the cervical enlargement and the pathological process underlying which was supposed to be always inflammatory. With his keen instinct for novel clinical types, Charcot[2] was among the first to recognize this affection, to give it an appropriate name, and to write an accurate description of it founded upon an autopsy. He called it *pachymeningitis cervicalis hypertrophica*, a name which indicates sufficiently both its nature and its seat. He was assisted in some of these observations by Joffroy, who subsequently wrote his thesis on this subject.[3] It is but just, however, to say that before the time of these observers Abercrombie and then Gull,[4] in England, had put on record similar cases,

[1] Read before the Philadelphia Neurological Society, March 26, 1894.
[2] Arch. de Physiologie, 1869 (with Joffroy).
[3] De la Pachymeningite Cervicale Hypertrophique. Paris, 1873.
[4] Guy's Hospital Reports.

but had failed to draw attention so accurately to their nature. Vulpian, subsequently, in his treatise on diseases of the nervous system, gave a concise description of this cervical affection, and later still, Burtin[5] wrote his thesis on spinal pachymeningitis, in which he described two types: first, the cervical, second, the lumbar. This cervical type of cord-lesion has become so well recognized now that most of the text-books describe it, although there are still a few that do not appear to recognize this affection, which can scarcely be called uncommon, and which presents some problems both in physiology and in pathology which give it quite an unique importance. These problems relate, first, to the physiology of the conduction of sensation; second, to the differential diagnosis between several pathological states, each of which while distinct in itself, can produce a complexus of symptoms very similar to that produced by each of the others.

The symptoms of pachymeningitis cervicalis may be divided into two groups. (1) Those pertaining to the level of the cervical region, and (2) those which are produced by ascending and descending degenerations. Among the first are neuralgic pains in the occiput, neck, shoulders and arms; paralysis, with muscular atrophy, in the shoulders, arms and hands; and stiffness of the neck with deformity of the spine. Among the second are spastic paraplegia, with increased knee-jerks and clonus, without muscular atrophy in the legs, and with or without (often without) paralysis of the bladder and rectum. There are in addition various alterations of sensation both at the level of and below the cervical lesion. These alterations were not duly studied in the earlier cases; in fact, in most of the papers no mention is made of alteration of the various modes of sensation. One of Charcot's[6] early papers is an exception, however, for in it there is express mention of thermo-anæsthesia as a symptom in one of his cases. It now seems certain that

[5] De la Pachymeningite Spinale. Paris, 1878.

[6] Charcot and Joffroy. Op. cit.

in the majority of these cases there is some alteration of sensation, and that this alteration may be the "dissociation symptom" described as peculiar to syringomyelia; *i. e.*, thermo-anæsthesia and analgesia without impairment necessarily of tactile sense, although this latter is occasionally modified in various areas. Other modifications, and combinations of modifications, of sensation will probably be found. Finally, there are sometimes trophic lesions in the bones and in the skin and nails, and there may be alterations in the pupils.

The pathology of pachymeningitis is familiar to those who have had opportunity to study lesions of the spinal cord.[7] As Vulpian describes this form of it, the cord is enlarged, filling the whole spinal canal in the cervical region. The enlargement is fusiform, and resistent on pressure, contrasting with the fluctuating sensation given by the cord in syringomyelia. The dura is much thickened and adherent, and the membranes separate from the cord with difficulty. The cord is sometimes fibrous, or it may be softened, and the gray and white matter cannot be distinguished easily. It contains no adventitious cavity as in syringomyelia. The nerve-roots are usually involved. As described by Vulpian and by Charcot in their own cases the appearances were not unlike those seen in tubercular pachymeningitis in the mid-dorsal region.

In presenting the brief histories of two patients this evening, I wish to emphasize the fact that both of them are traumatic cases, and both in men past the prime of life who had previously been in good health. It has been rather too much the custom in the past to regard cold as the most puissant agent in the production of all forms of pachymeningitis. That pre-eminence will probably be held henceforth by infection and by trauma. In presenting these cases, moreover, I purposely have called them simply examples of traumatic affections of the cervical cord, and I do not intend to commit myself to a

[7] See a paper, with illustrations, on "Pachymeningitis and Myelitis from Pott's Disease, etc.," by the writer, *University Medical Magazine*, Dec., '93.

statement that they are necessarily, or even probably, examples of an inflammatory process. They simply conform to a clinical type, due, in their cases, to trauma, and I confess that I am not satisfied in my own mind that they are not degenerative rather than inflammatory.

James G., aged 55, white, male, stonecutter by trade; gives a negative family history. He has no personal history of syphilis or alcoholism.

In 1876, he fell from a scaffolding in front of the U. S. Mint in this city, striking the back of his neck. A fracture of the spinal column was diagnosticated. The entire body, including both arms and legs, was paralyzed; breathing was embarrassed, and urination, but not defecation, was affected. It is reported that there was no marked disturbance of sensation below the waist, but the arms were anæsthetic. In three months he regained power in the legs; the arms did not improve so quickly, the return of power in them being delayed for eight months. He was weak for over two years; the neck was deformed; the right leg a little feebler than the left. In three years he returned to his work as a stonemason, claiming now that he was as strong then and as able to do hard work as ever. This return to work is a very significant incident in the history of the case. In 1884, five years later, he fell again, this time from a ladder, a distance of fifteen feet, and struck the back of his head and his spine between the shoulders. He was unconscious for six hours. This fall was followed by a return of the paralysis of the legs and arms, loss of control over the bladder, and obstinate constipation. According to his statement there was no marked loss of sensation, but we do not know how carefully that was determined. There was no priapism. There was severe pain at the seat of injury, and headache for three months. Power returned to the legs in two months, in the arms slightly at the end of seven months. After confinement to bed for four months, the patient began to get about, but he has not been able to do any work since the second accident.

The present condition is as follows: The patient has advanced atrophy of the shoulder-muscles of both sides, including the deltoid, pectoralis major, infra and supraspinati and the lower part of the trapezius; also of both the arm and forearm groups of muscles, and of almost

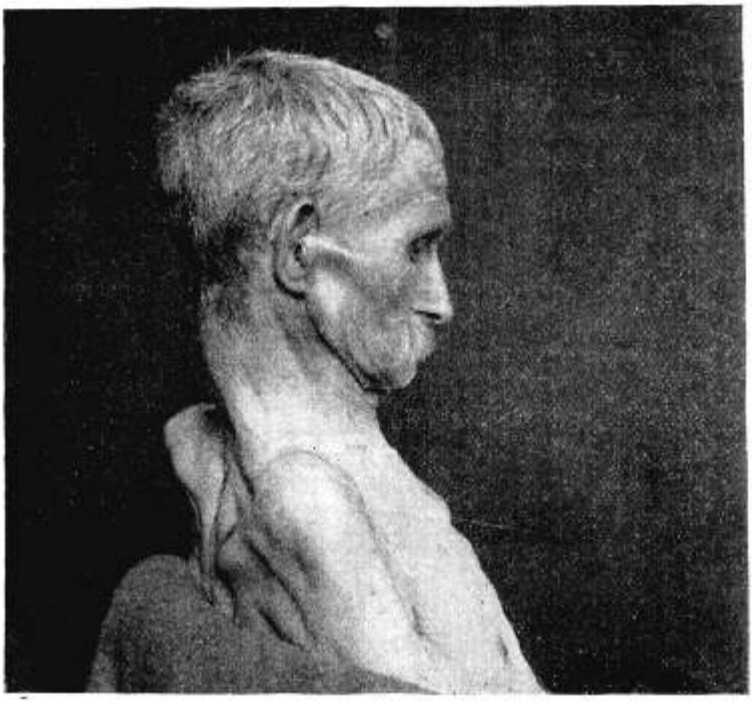


FIG. 1. A Case of Trauma of the Cervical Region of the Spinal Cord.

all the muscles of the hands. (See photograph, Fig. 1.) The thumb is in the position of extension of the metacarpal bone, flexion of the proximal and extension of the distal phalanx—not exactly the position assumed in "main-en-griffe," which position of the hand is not present here, although what seems to be a commencement of it is seen in the middle and ring fingers. Fibrillary contractions are well marked in some of the wasted muscles. The myotatic irritability is much increased, especially in the biceps and in the extensors of the wrist. There is a little power of flexion and extension of the forearm, but except for this, the arms are quite paralyzed and useless. The muscles respond to both currents; there is some decrease in the most wasted muscles, and some hyperexcitability, with duration tetany, in others.

The patient has not head-drop.

The legs are paretic and slightly contractured. The knee-jerks and ankle-clonus are very lively. There is no muscular atrophy in the legs. There are no trophic lesions, no sympathetic lesions, no bulbar symptoms, no disturbance of respiration, nor any paralysis of the bladder. There is marked stiffness of the neck, with deformity of the cervical spine. This deformity is a hump-like deviation of the spine, such as may have been caused by a fracture. There is also slight scoliosis. There is still more or less neuralgic pain about the neck and the occiput.

The sensory tests in this case are of great interest. (Figs. 2 and 3.)

Tactile sensation is good everywhere; repeated tests have always demonstrated this.

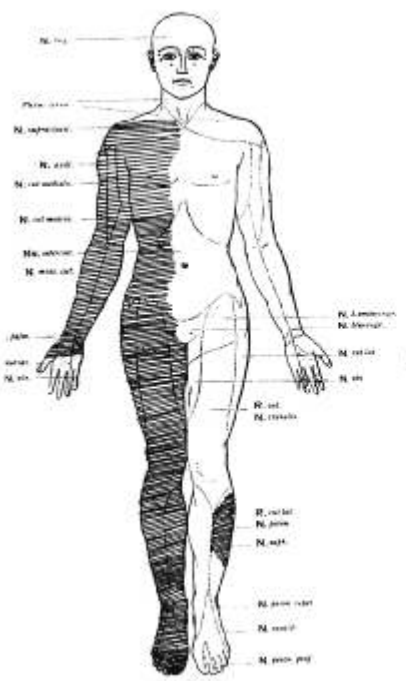


FIG. 2.

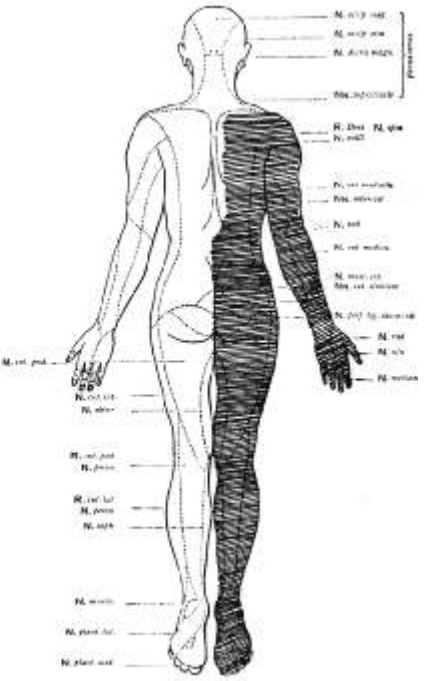


FIG. 3.

FIG. 2. Thermo-Anæsthesia and Analgesia on right side. Tactile Anæsthesia in small area on left leg.

FIG. 3. Thermo-Anæsthesia and Analgesia on right side. Tactile Anæsthesia in small area on left leg.

Thermo-anæsthesia is marked on the right side, as shown in the schemata. It is more properly an anæsthesia to cold. The patient distinguishes heat in the affected areas, but everywhere in these areas calls cold hot. There is analgesia quite marked in some of the affected areas. This condition has practically remained unchanged for a year, very recent tests corresponding closely to those made one year ago.

It is thus seen that this patient presents a very close resemblance to a case of syringomyelia in the cervical enlargement. The motor and sensory lesions, their location and distribution, are closely analogous. Trophic and vasomotor lesions are alone wanting. The question, indeed, presents itself, whether the lesion is a true syringomyelia of traumatic origin. Bruhl,[8] in his monograph, recognizes trauma as a cause of syringomyelia, and several of his reported cases illustrate this etiology. One of Charcot's latest lectures[9] was on a case of cervical trauma, caused by a gun-shot, simulating syringomyelia. The injury was probably to one of the nerve-roots, and thermo-anæsthesia was a marked symptom. In our case, an autopsy alone, it seems to me, could determine the exact pathology.

The second case is briefly as follows:

Patrick C., aged 60 years; white, male, laborer; gives a negative family history. No syphilis. Thirty years ago, he was injured in the cervical and dorsal spine, by falling into a canal and being jammed between two canal boats. For treatment he was kept strapped to a long dorsal splint or platform for five weeks. In about three months he had so far recovered, that he returned to work, and continued to do hard work as a common laborer up to the time of his second accident. The second accident happened one year before his admission to the hospital, about three years ago. He was in good health at the time. While working with others digging out a clay-bank, part of the bank fell and buried him. He was under the slide for about one-half hour before he was rescued. He was unconscious and remained so for

[8] Contribution a l'étude de la Syringomyélie. Paris, 1890.

[9] Clinique des Maladies du Système Nerveux, Paris, 1892, p. 333.

twenty-four hours. On recovering consciousness, he was found to be paralyzed in the left arm and leg. The face was not involved, and there was no affection of speech. He was in the hospital for the accident about six months. Contractures gradually began in the affected arm and leg.

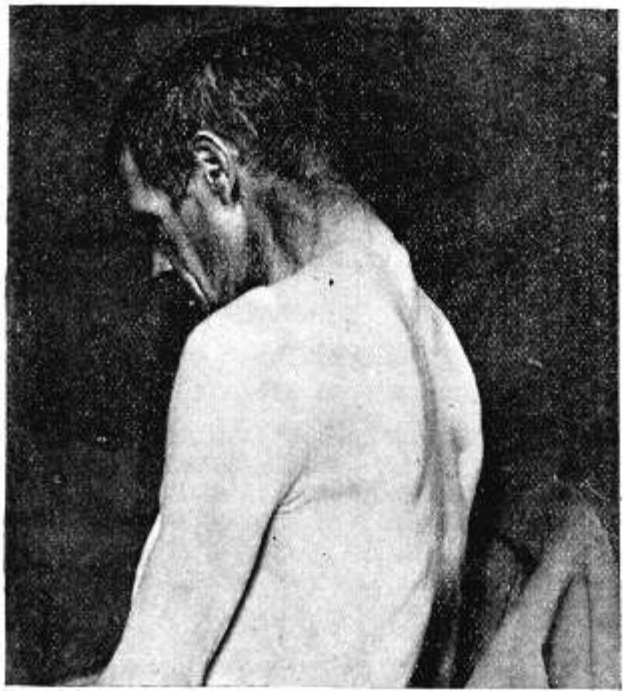


FIG. 4. A Case of Trauma of the Cervical region of the Spinal Cord.

His present condition is as follows: He has a marked angular deformity of the first dorsal spine, and less markedly of the second dorsal and of the seventh cervical. There is a depression also in the region of the lumbar vertebræ. The patient carries his head in a set position due to the stiffness and deformity in his neck; his chin almost rests upon his thorax (see photograph Fig. 4). There is paralysis decidedly more marked in the left arm and leg than in the right, with contracture of the hand and forearm. The shoulder-muscles, especially the infra and supra-spinati, are atrophied, as are also the

muscles of the arm; and there are fibrillary contractions in them. Clonus is marked in the fingers, wrist, and ankle of both sides. The phenomena of so-called "spinal epilepsy" are readily excited in the legs. This general leg-clonus, once started, will continue almost indefinitely.

The patient is unable to distinguish heat from cold, or to recognize either in the region marked on the chart. It will be observed that this thermo-anæsthesia is on the right side, or the side opposite the paralysis of motion. (Figs. 5 and 6.) There is an area of anæsthesia involving the right leg. (Fig. 7.) The man complains much, however, of a form of hyperæsthesia or pain, and paræsthesia of the right, or unparalyzed, side. He experiences constantly a sensation of burning on this side, and is in

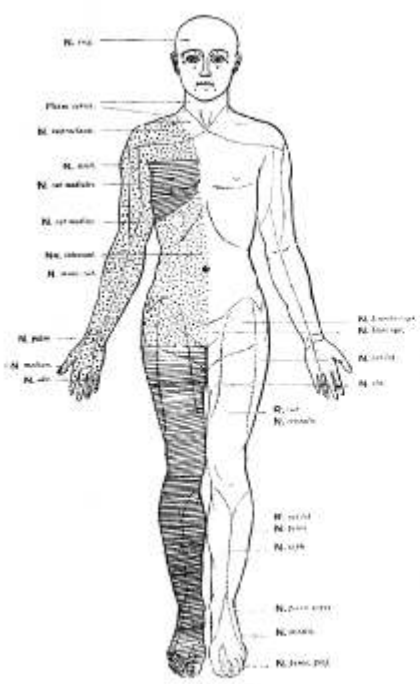


FIG. 5.

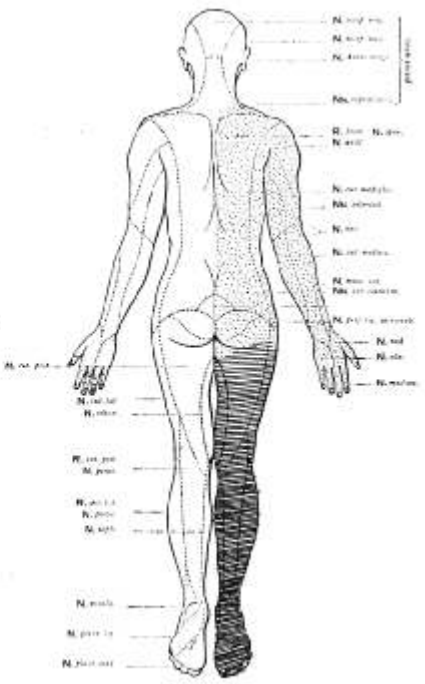


FIG. 6.

FIG. 5. Thermo-Anæsthesia indicated by cross-lines. Analgesia in the same areas, and also in areas indicated by dotted shading.

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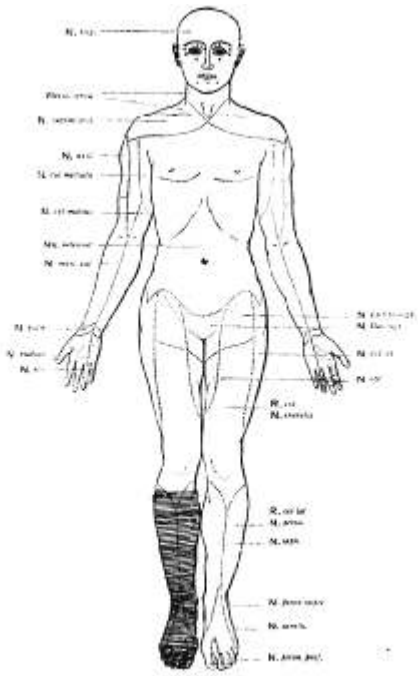


FIG. 7. Tactile Anæsthesia, varied with delayed sensation.

the habit of putting cold water on it to allay the heat, but he finds this remedy ineffectual—an interesting fact when it is recalled that he cannot distinguish cold from heat.

He is profoundly analgesic in the right arm and leg.

There is no involvement of the iris; no impairment of respiration, nor any interference with the innervation of the bladder.

It will be observed that the prominent feature in this case is its hemiplegic type. This is so pronounced that a hasty observer might be inclined to consider the case one of cerebral lesion. This view is opposed, however, by the history of the case, the direct effect of accident, the deformity of the spine, the absence of paralyses of the face or tongue, the sensory changes, and the atrophy and fibrillation in the paralyzed muscles. It follows closely the Brown-Sequard type—the paralysis of motion being on one side, the thermo-anæsthesia on the other. Its resemblance to syringomyelia is not so striking as that of the first case, yet it must be borne in mind that

there is an hemiplegic type of that disease. I once saw in my service at the Philadelphia Hospital a strict hemiplegia, the result of a lesion in the cervical cord, and some years ago, with Dr. Deaver, reported the case, in which the latter performed a laminectomy.

THE PHYSIOLOGY OF THE CONDUCTION OF SENSATION.

The hopeless confusion that has prevailed among physiologists upon the subject of the sensory paths in the spinal cord is known to all students. Their opinions are diametrically opposed to each other, and most of them are being contradicted by more recent and improved research. Among these opinions are the following[10]: That sensory impulses are transmitted by the lateral column of the opposite side; that they are transmitted by the lateral columns of the same side; that they are transmitted by the posterior columns of both sides; and, finally, that they are transmitted by the gray matter. Almost all of these statements are probably erroneous, unless amplified or modified as the case may be. The dictum of Brown-Sequard especially, that a hemi-section of the cord causes anæsthesia below the lesion on the opposite side of the body, a dictum that has been received unreservedly by neurologists for years past, must be regarded henceforth with grave suspicion. The causes for uncertainty and confusion have been of three kinds; the first has been the inability to control the secondary results of wounds before the days of aseptic surgery; the second has been the reliance placed entirely upon sensory responses in animals; the third has been the defect in histological study. All of these causes have either been removed or controlled to some extent. The methods of aseptic surgery have removed at least one element that formerly confused and even vitiated the results of experimenters.

The exact knowledge which we possess on the subject of sensory conduction in the cord is still limited, and when kept free from speculation and hypothesis

[10] Quoted from Gotch. "Recent Research on the Spinal Cord." *Liverpool Medico-Chirurgical Journal*, vol. xiii. 1893.

may be summarized very briefly. It is derived from three sources: (1) From histology. (2) From experiment. (3) From disease.

Histology.—According to Ramon y. Cahal, the sensory nerve-fibre, entering by the posterior root from its cell of origin in the spinal ganglion, divides dichotomously (or in half) in the postero-external column, one branch passing upwards in the postero-internal column, the other downwards. From these longitudinal branches there arise short transverse branches, which penetrate the gray matter, and there end in the tufts or "arborisations," which characterize the peripheral or distributory ends of nerve-filaments. This course is proved further by the fact that if a posterior nerve root is cut the ascending degeneration is seen only in the posterior column of the same side (Gotch). It is promptly checked in the gray matter, where the terminal arborisations occur, and no ascending degeneration is seen anywhere on the opposite side (Gotch). The ascending degeneration ceases after travelling up the posterio-internal column in the nuclei of the funiculus gracilis and funiculus cuneatus, which are ganglionic bodies, apparently beginning another stage of the sensory pathway. This view of the histology of the entering sensory nerve-fibre is adopted by Piersol, by Stirling and by Köllicker. In its simplicity it is very striking, and it leaves little room for the hypothesis that there are separate strands for tactile, thermal and painful impressions, at least in the posterior root. Certainly these impressions all enter the cord over identical fibres. It is lacking, absolutely, as far as it goes, in any proof of a sensory decussation low down in the cord. These tufts or arborisations, in which the sensory fibre ends in the gray matter, are apparently in close contact, but not in direct anatomical relation, with the ganglion cells, both in the posterior and anterior horns and in Clark's column, and from these ganglion-cells other nerve fibres are projected, the course of which, except those from Clark's column, which pass over to form the ascending cerebellar tract,

and those which pass out the anterior roots, is not very clear. Some of them pass up the antero-lateral columns, others decussate through the gray commissure with the elements of the opposite side; but the evidence from histology seems to be that these decussating fibres are relatively few, *i. e.*, in very small numerical proportion to that of the others. The contrast with the decussating fibres from the anterior horns, which run so conspicuously through the white commissure in front, is very striking.[11] Finally, it may be borne in mind that there is a very rich decussation of sensory fibres in the decussation of the fillet in the medulla oblongata, and that these are in direct line from the fibres in the posterior columns of the cord to the brain above.

Experiment.—Gotch [12] in his recent paper on the spinal cord makes the explicit statement that "recent physiological research shows that, in opposition to the views formerly advocated by many neurologists, the path for sensory conduction is almost entirely on the same side as that of the entering sensory nerves." Mott showed that the monkey after hemisection of his cord would remove a clip from the skin of the leg opposite the lesion, but would allow the one on the leg on the side of the lesion to remain. In the experiments of Gotch and Horsley the sensory tests, which are always uncertain in animals, were dispensed with, and the capillary electrometer was used instead. This instrument registers the passage of a nerve-current, and in their experiments seemed to show that mild stimuli send impulses up the posterior columns of the same side. These are the paths of least resistence. These mild stimuli represent probably simple tactile impressions, passing up the most direct way; *i. e.*, by the posterior columns. More violent

[11] If the evidence from histology is to be accepted there is as much, or even more, reason to say that the motor fibres decussate at each level of the cord as there is to claim that the sensory fibres so decussate. The truth probably is that all these fibres, in both the anterior and posterior commissures, are purely commissural.

[12] *Op. cit.*

impressions, however, such as would be made by heat, cold and pain, radiate through into the gray matter of the cord. Here they have a path of greater resistance (as we have seen histologically, a path not of direct continuity, but through the tufts of fibrils of the sensory fibres, hence passing in some way to the cells of the gray matter), and through the gray matter they possibly emerge again into the white conducting tracts, even of the lateral, and possibly of the opposite, posterior column. If accurate, this would mean that pain and painful temperature sensations are only of different degree, and hence excite a wider and more complex nervous mechanism than simple touch. From the clinical standpoint, the fact of separate alterations of the various modes of sensation by different diseases might thus be explained.

Disease.—In this connection I desire simply to call attention briefly to the fact that not only diseases involving extensively the gray matter, as syringomyelia, cause alterations in the temperature sense, but also that diseases of peripheral parts, as pachymeningitis and neuritis, may also cause them. The patients whose cases I report to-night are possibly mixed cases of injury both to the cord and to the nerve-roots.

An Addition to the Etiology of Degenerative Changes in the Cord.—Dr. Fr. Tuczek, in the *Prager Medicinische Wochenschrift*, gives an interesting account of some recent investigations of his to determine the possible relation between ergotism and dementia paralytica. One fact brought to notice was the association of pellagra in patients who were suffering from nervous symptoms induced by ergot. Histological examination of the nervous system in cases where death had ensued, with pellagra present, revealed marked degenerative changes in the spinal cord, especially in the posterior and lateral columns. The author believed that his studies were sufficiently confirmative to make the statement that the poison from the diseased grain acted in such a manner upon the nervous system, as to produce degenerative changes in the spinal cord, and that an important addition was thereby made to the etiology of cord disease. B. M.